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Review

Acidification Reshapes Zooplankton Nutrition from Physiological Costs to Trophic Transfer: A Review

Xin Liu [1,*], Huanan Gao [2], Yasushi Iseri [3], Qiang Huang [1,4,5], Peixue Song [6] and Aimin Hao [3,*]

1 Guangxi Key Laboratory of Marine Environmental Science, Guangxi Academy of Marine Sciences, Guangxi Academy of Sciences, Nanning 530007, China; huangqiang@gxas.cn
2 Hainan Academy of Ocean and Fisheries Sciences, Haikou 570100, China; gaohn2015@gmail.com
3 College of Life and Environmental Science, Wenzhou University, Wenzhou 325035, China; iseri@wzu.edu.cn
4 Beibu Gulf Marine Industry Research Institute, Fangchenggang 538000, China
5 Guangxi Laboratory of Oceanography, Nanning 530007, China
6 School of Urban and Architectural Engineering, Zaozhuang University, Zaozhuang 277160, China; songpeixue1990@163.com
* Correspondence: author: lx@gxas.cn (X.L.); hao_aimin@wzu.edu.cn (A.H.)

Simple Summary

Zooplankton are small drifting animals in oceans, lakes and rivers, and are an important food source for fish. Acidification of aquatic environments may weaken this food web link. This review combines marine and freshwater studies to show how acidification affects zooplankton growth, reproduction and value as prey. Acidification may act directly through physiological stress and indirectly by changing food quantity and quality. Zooplankton may survive but grow more slowly, use more energy or produce fewer offspring, and these responses depend on food, life stage, temperature and other stressors. Some species can adjust, but often at an energetic or reproductive cost. We recommend measuring feeding, energy use, growth and reproduction together to detect hidden effects on aquatic food webs and fisheries.

Abstract

Environmental acidification can alter zooplankton biochemical composition and production before short-term mortality becomes detectable. This narrative review synthesizes marine and freshwater evidence through a resource budget framework linking food acquisition, assimilation, maintenance expenditure, growth, reproduction and trophic transfer. Direct effects involve altered feeding, ion and acid–base regulation, respiration and cellular stress. Food-mediated effects arise from changes in prey abundance, composition, stoichiometry and essential compounds, together with consumers' capacity to assimilate or compensate for altered diets. Experiments separating prey and consumer exposure best distinguish these pathways. Maintained survival or adult reproduction can coexist with delayed development, reserve depletion or reduced recruitment. Responses depend on food supply, developmental stage, seasonal strategy, exposure variability and interacting stressors. Cross-generational and lake recovery studies reveal the limits and costs of physiological buffering, adaptation and ecological re-establishment. Effects on higher consumers remain less directly resolved than changes in zooplankton condition. We identify measurements needed to distinguish reduced intake from increased maintenance costs and quantify the amount and nutritional quality of production reaching predators. This framework organizes heterogeneous outcomes by mechanism while retaining each study's chemical and ecological boundaries.

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1. Introduction

Acidification is a global environmental stressor affecting both marine and freshwater ecosystems, although it arises through different mechanisms and has different chemical consequences in each system [1,2]. Ocean acidification changes carbonate chemistry through the uptake of anthropogenic carbon dioxide (CO_2), increasing acidity and reducing carbonate availability [1,3]. Coastal metabolism, eutrophication and transport modify this background trend [4–6]. Copernicus-based estimates indicate that the global mean surface seawater pH decreased from 8.11 in 1985 to 8.04 in 2024 (Figure 1). This decline corresponds to approximately 18% more hydrogen ions [7]. Future exposure diverges among Shared Socioeconomic Pathways (SSPs). A 14-model Earth system ensemble constrained by observations projected that the global mean surface pH would decline by 0.39, to 7.68, by the end of the 21st century under SSP5-8.5 [8]. Idealized simulations also show that Arctic aragonite-corrosive conditions can persist during atmospheric CO_2 decline, indicating delayed recovery [9].

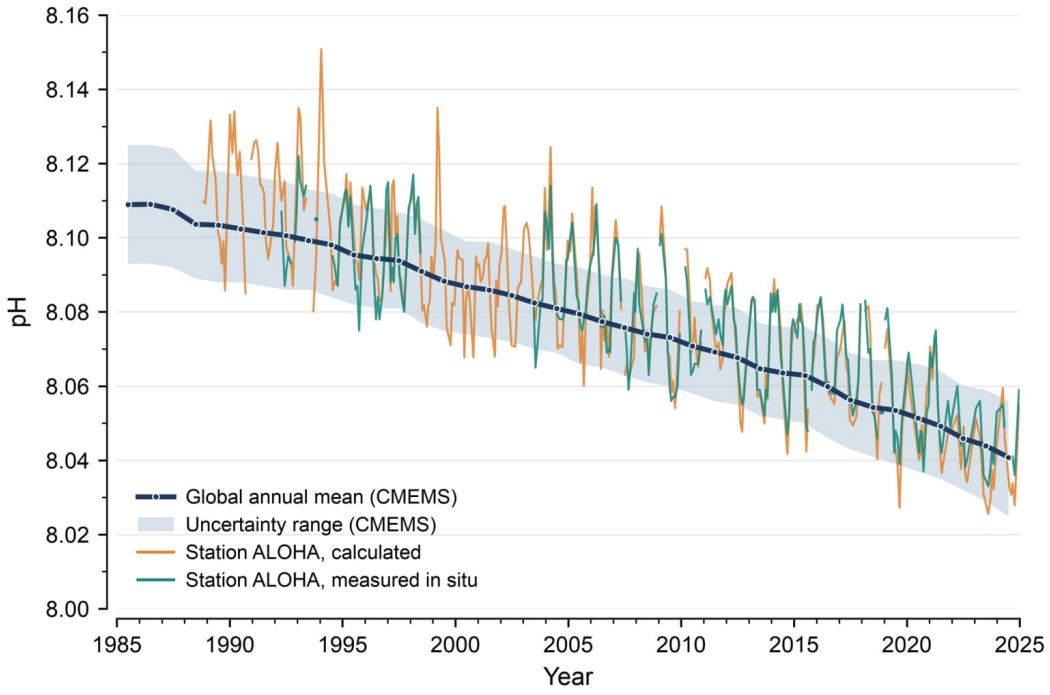


Figure 1. Decline in ocean pH measured at the Aloha station and yearly mean surface seawater pH reported on a global scale. Data referred to European Environment Agency [7]. Aloha station: Data originate from the ALOHA station pH time series in the central North Pacific, one of the world's longest continuous ocean acidification monitoring records. CMEMS: Global mean surface ocean pH from the Copernicus Marine Service, based on a reconstruction approach combining in situ observations, satellite-derived data and empirical relationships. The indicator is available annually from 1985 to 2024. Trend and uncertainty are calculated as the slope and residual standard deviation of a linear least squares regression.

Freshwater acidification involves acid deposition, catchment processes and CO_2 enrichment, with buffering capacity and hydrology governing regional exposure [2,10]. Emission controls have improved water chemistry, but calcium depletion and persistent community

changes can delay biological recovery [11,12]. Long-term acid deposition continues to affect lake chemistry in southwest China [13]. Catchment processes can also cause acidification. In a central Chinese lake, groundwater inputs with proportionally more dissolved inorganic carbon (DIC) than alkalinity contributed to seasonal acidification, and lake water pH ranged from 7.26 to 8.83 among observations [14]. In contrast to these observed changes, future CO_2-driven acidification of lakes has been estimated through model projections. Models for 18 northeastern United States lakes projected mean pH declines of 0.15 and 0.32 from sampled lake conditions by the end of this century under scenarios assuming atmospheric CO_2 concentrations of 600 and 1100 ppm, respectively [15]. Additionally, acidification must also be considered alongside global warming because temperature alters metabolic demand and the biological consequences of acid–base disturbances [16,17]. Acidification exposure therefore differs among systems in chemical pathway, magnitude and timing, and its biological consequences are further modified by temperature, season and resource conditions.

Zooplankton connect environmental pressures to food web functioning through grazing, prey production, nutrient recycling and carbon export [18–20]. Cladoceran and copepod zooplankton regulate phytoplankton through grazing, while they link lower trophic levels to fish production [18–20]. Direct acidic exposure can change swimming, encounter rates and prey selection before measurable changes in body composition occur. In a seasonally hypoxic and acidified fjord, copepods swam more slowly and made fewer escape jumps [21]. Their ecological functions depend on both the quantity and biochemical quality of the biomass they produce. At natural CO_2 seeps, lower *Labidocera* abundance was not explained by measured feeding or egg development [22]. Their central food web roles make vulnerability to acidification ecologically consequential. Ion and acid–base regulation provide physiological links between external acidity and resource allocation [23,24]. Previous studies identified depth-specific distributions determine exposure, as shown for Northeast Pacific pteropods [25], and reduced chemically suitable habitat for sensitive polar pteropods, as aragonite saturation declined [26]. Such variability strengthens the need to identify vulnerable taxa and mechanisms under ecologically relevant conditions.

Understanding these vulnerabilities requires linking acidification to zooplankton production and trophic transfer. Growth or recruitment may decline due to reduced food quality, lower food intake, or increased maintenance costs, mechanisms that cannot be distinguished based on survival or abundance alone. This narrative review synthesizes marine and freshwater evidence, emphasizing crustacean zooplankton, through a nutritional resource budget framework. This review builds on established concepts of energy allocation, including dynamic energy budget theory, scope for growth and energy-limited stress tolerance, as well as ecological stoichiometry. Its contribution lies in placing direct physiological costs and food-mediated changes in prey quality within a single budget, so that similar reductions in growth or reproduction can be traced to different budget components. We distinguish direct physiological effects from food-mediated effects and link resource allocation to life history traits and trophic outcomes. We also examine how interacting stressors, adaptation, and recovery influence vulnerability and identify key measurements for quantifying the quantity and quality of production available to higher trophic levels. Similar exposures can produce contrasting outcomes because responses depend on which budget component is affected and whether food supply allows compensation.

In this review, relevant studies were identified through searches of Google Scholar conducted from January 2025, with a final update in October 2026 using combinations of terms for acidification (e.g., acidification, CO_2, pCO_2, low pH, acid deposition), zooplankton (e.g., copepod, cladoceran and rotifer) and nutritional or ecological responses (e.g., food quality, fatty acid, stoichiometry, energy budget, reproduction and trophic transfer),

supplemented by screening the reference lists of key studies and reviews. For example, searches combined "acidification" with copepod and with "fatty acid" or "food quality"; pCO_2 or "low pH" with zooplankton and with "energy budget" or reproduction; and "acid deposition" or "lake acidification" with cladoceran or rotifer. The cited studies range from 1974 to 2026, with earlier freshwater studies documenting acid deposition and recovery. Titles and abstracts were first screened for relevance, and full texts were then assessed. We prioritized studies that reported exposure conditions together with physiological, nutritional, life history, community or trophic responses, and retained studies reporting neutral and positive responses alongside those reporting adverse effects.

2. Exposure Chemistry

Acidification refers to a sustained decrease in pH and the associated change in acid–base chemistry, irrespective of its cause. In the open ocean, acidification is driven mainly by CO_2 uptake, whereas coastal waters and freshwaters involve additional pathways. Freshwater acidification is used as a general term that includes acid deposition, catchment processes and CO_2 enrichment. Acid deposition, the atmospheric input of sulfuric and nitric acids, is therefore treated as one pathway of freshwater acidification [10,27]. CO_2 enrichment refers to increased dissolved CO_2 and dissolved inorganic carbon (DIC) from atmospheric uptake, respiration or groundwater inputs [2,28]. Low pH is used only to describe a measured exposure condition, because similar pH values can result from different chemical pathways, and CO_2-driven changes in carbonate chemistry are not necessarily proportional to the change in pH [5,6]. Buffering capacity is used as a general term for the capacity of water to resist pH change. In seawater, it is governed mainly by total alkalinity (TA) and the carbonate system [29]. In freshwaters, this capacity is quantified operationally as acid-neutralizing capacity (ANC), the ability to neutralize strong acid inputs, which is related to but not identical with carbonate alkalinity [30].

Exposure chemistry encompasses the processes that generate acidity and the chemical conditions encountered by zooplankton. In marine ecosystems, CO_2 uptake increases DIC without altering TA, assuming no other biogeochemical reactions occur. Redistribution among dissolved CO_2, bicarbonate and carbonate increases hydrogen ion availability, lowers pH and reduces carbonate mineral saturation [1,3]. DIC and TA therefore provide complementary information, as pH alone cannot distinguish whether acidification results from CO_2 accumulation, acid production, or mixing between water masses [5,6]. These underlying processes must be distinguished before acidification exposures among different habitats or experimental treatments can be meaningfully compared.

Coastal and estuarine chemistry reflects local processes superimposed on atmospheric CO_2 uptake. Organic matter respiration produces CO_2 while consuming oxygen, allowing eutrophication to intensify subsurface acidification [4]. Redox reactions can also change TA. In Chesapeake Bay, the oxidation of reduced compounds, including hydrogen sulfide, contributed to acidification near the oxic–anoxic interface, while river–ocean mixing and limited buffering further influenced the observed pH minimum [5]. Riverine inputs, upwelling and ecosystem metabolism consequently modify both the magnitude of and variability in coastal exposure [6]. Decadal observations in a eutrophic subtropical coastal area likewise link acidification with nutrient and ecosystem processes [31]. Low pH should therefore be characterized alongside oxygen and the local carbon–alkalinity balance.

In freshwaters, acid deposition and catchment processes can alter ANC, base cation concentrations and metal chemistry [10,32]. Sulfate and nitrate inputs must be interpreted together with neutralizing cations and their processing within catchments, rather than treated as equivalent acid loads [13,27,33]. Consumption of alkalinity during acid neutralization can reveal changing buffering conditions even when pH varies little [33]. Naturally

occurring organic acids provide an additional source of acidity, making the distinction between natural and anthropogenic acidification important in humic waters [34]. Acidification can be accompanied by calcium depletion and changes in aluminum availability, resulting in substantial differences in ionic conditions among waters with similar pH values [11,23,32]. A comprehensive assessment of freshwater acidification exposure therefore requires measurements of ANC or alkalinity, major ions, dissolved organic carbon (DOC) and relevant metals, in addition to pH.

CO_2 enrichment also represents an important pathway of acidification in freshwater ecosystems. Great Lakes carbonate system scenarios show that rising atmospheric CO_2 can lower lake pH without requiring increased acid deposition, while natural variability can complicate detection of the trend [2]. Groundwater discharge may supply disproportionately more DIC than alkalinity and thereby promote seasonal lake acidification [14]. CO_2-driven pH change can also alter aluminum speciation under particular lake conditions [15]. These processes highlight the distinct chemical effects of CO_2 enrichment and strong acid addition: CO_2 enrichment primarily alters inorganic carbon speciation, whereas strong acid addition reduces alkalinity [2,33]. Experimental freshwater CO_2 exposures and acute acid stress assays should therefore be compared based on their measured water chemistry and experimental manipulation methods, rather than solely on target pH values [35,36].

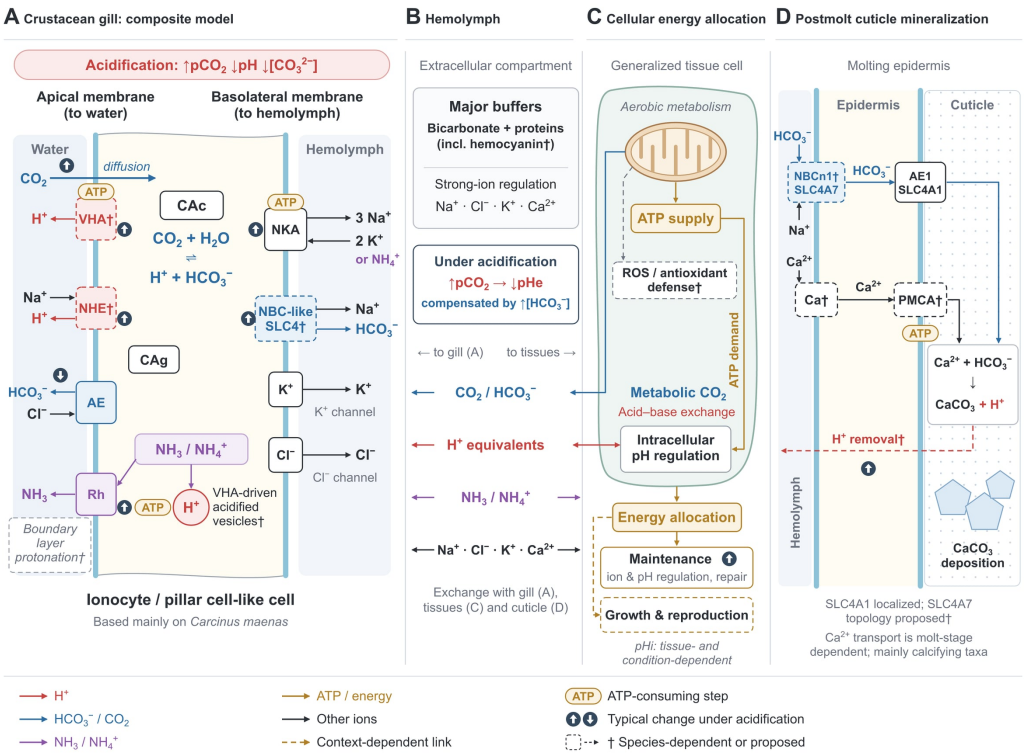
Acidic exposure also varies with depth, transport and season. Natural pCO_2 gradients under Arctic winter sea ice demonstrate that copepods can occupy chemically distinct water masses [37]. Depth-resolved distributions of *Limacina helicina* demonstrate how assuming a vertically uniform distribution can lead to inaccurate estimates of exposure to low aragonite saturation states [25]. In freshwater catchments, acidic snowmelt can cause episodic acidification, but the pH of meltwater cannot be directly extrapolated to receiving lake waters [38]. Future Arctic carbonate system projections similarly describe scenarios rather than measured exposure of every organism [39]. The relevant chemical history therefore includes the duration, frequency and sequence of low-pH conditions within the occupied habitat, together with any covarying oxygen, temperature or ionic changes.

Marine carbonate chemistry should be constrained by at least two independently measured carbonate system parameters, such as pH and TA, with temperature, salinity and the calculation conventions stated [1,37,40,41]. The choice of pH scale is essential, as copepod studies reporting pH on both National Bureau of Standards and total scales demonstrate that numerical pH values depend on the scale used [41]. Freshwater studies should identify the acid source or CO_2 manipulation and accompanying buffering and ionic conditions. Carbonate calculations require assumptions appropriate to freshwater ionic composition [2]. Across both marine and freshwater systems, initial and time-resolved measurements should be used to determine whether experimental treatments reflect present-day conditions, episodic extremes, or future scenarios. This chemical characterization provides the necessary context for interpreting biological responses as below.

Although the chemical drivers differ, the resource budget framework can be applied to both marine and freshwater systems because acidification in each case can alter resource acquisition, maintenance expenditure and allocation to growth and reproduction. However, the dominant maintenance costs are likely to differ. Under marine CO_2-driven acidification, costs arise mainly from acid–base regulation and, in calcifying taxa, from reduced carbonate saturation [24,42], whereas in acidified freshwaters, low pH is often accompanied by low calcium and elevated aluminum, which increase ion loss and impair ion uptake [11,23,43].

3. Direct Physiological Effects on Zooplankton Nutrition

Exposure to acidification conditions can directly impose physiological demands on zooplankton. Their nutritional consequences depend on whether maintaining internal conditions alters energy expenditure or the resources available for growth and reproduction. Figure 2 organizes epithelial transport, systemic buffering, nitrogen excretion and mineral balance into a mechanistic framework [42]. Because many of these mechanisms were characterized in larger crustaceans, they are presented as hypotheses to be tested in zooplankton unless direct evidence is available.



VHA, V-type H⁺-ATPase; **NHE**, Na⁺/H⁺ exchanger; **NKA**, Na⁺/K⁺-ATPase; **NBC**, Na⁺/HCO_3^- cotransporter; **AE**, anion exchanger; **Rh**, Rhesus-like protein; **CAc**/**CAg**, cytosolic/membrane-bound carbonic anhydrase; **PMCA**, plasma-membrane Ca^{2+}-ATPase; **Ca†**, Ca^{2+} entry pathway; **SLC4**, solute carrier family 4; **pHe**/**pHi**, extra-/intracellular pH; **ROS**, reactive oxygen species.

Figure 2. Composite model of crustacean physiological regulation and cellular energy allocation. (**A**) Branchial ion transport, acid–base regulation and ammonia excretion. (**B**) Hemolymph buffering and systemic exchange. (**C**) Intracellular pH regulation, ATP supply and demand, and energy allocation to maintenance, growth and reproduction. (**D**) Calcium and bicarbonate transport supporting postmolt cuticle mineralization. The model integrates mechanisms across crustaceans rather than representing a single species or condition. Solid connections depict established physiological relationships; dashed outlines or arrows and daggers indicate species-dependent or proposed components. Dashed gold connections indicate context-dependent links to growth and reproduction. Circled arrows indicate the typical direction of change under acidification, and ATP labels mark ATP-consuming steps.

3.1. Ion and Acid–Base Regulation

Epithelial ion transport provides the first connection between external chemistry and internal regulation. Basolateral Na^+/K^+-ATPase maintains electrochemical gradients, while carbonic anhydrases couple CO_2 hydration to H^+ secretion and HCO_3^- handling. V-type H^+-ATPase, exchangers, bicarbonate transporters and channels contribute differently across species, salinities and epithelial regions [44–48]. These differences preclude assuming an identical transport response across zooplankton taxa. Direct evidence in *Daphnia magna* shows that ambient Ca^{2+} modifies the effects of low pH on Na^+ influx and whole-body

sodium [43]. Ionic composition therefore helps determine the regulatory challenge associated with a given external pH.

Epithelial exchange operates alongside buffering, gas exchange and nitrogen excretion. Hemolymph bicarbonate and proteins help buffer acid–base disturbances [49], while intracellular pH regulation depends on tissue-specific ion exchange [50,51]. Ventilation and circulation also coordinate gas exchange and physiological demand [52]. Branchial excretion links this regulation to the removal of metabolic products. In the green crab, V-type H^+-ATPase and microtubule-dependent processes participate in ammonia excretion [53]. An apical Rhesus-like glycoprotein provides a route for both ammonia and CO_2 transport [54]. These mechanisms explain how internal conditions can be maintained, but their energetic cost under acidification requires measurements in the focal organism.

Mineral balance introduces an additional regulatory demand during the molt cycle. Ca^{2+} transport and bicarbonate delivery must be coordinated with cuticle resorption and deposition [55,56]. SLC4 family bicarbonate transporters have been implicated in crustacean mineralization [56]. Excretory organs provide another route for modifying internal chemistry. Antennal glands also contribute to acid–base and nitrogen balance by regulating sodium, ammonia and inorganic carbon fluxes, although the direction and magnitude of these fluxes differ among taxa and environments [57–59]. Evidence from larger crustaceans identifies mechanisms worth testing in zooplankton. Establishing their nutritional consequences requires linking regulation to expenditure, reserve use and production, rather than inferring costs from anatomy alone.

3.2. Cellular Metabolism and Oxidative Stress

Cellular measurements can reveal the metabolic adjustments accompanying physiological compensation. Metabolomic analysis of *Calanus glacialis* detected stage-specific changes in ATP-related metabolites, tricarboxylic acid cycle intermediates, and amino acid pathways [60]. These patterns indicate metabolic reorganization, but metabolite profiles alone cannot quantify energy flux or the resources available for production. Food and reserve status also affect their interpretation. In food-deprived *Calanus* spp., five days of starvation produced greater metabolic stress than the tested warming and acidification treatments [20]. Exposure effects therefore need to be evaluated within the nutritional conditions under which cellular responses were measured.

Oxidative responses provide a complementary indication of cellular stress, especially when linked to functional outcomes. In *Tigriopus japonicus*, low pH altered reactive oxygen species and antioxidant activity alongside prolonged development and reduced size and fecundity [61]. This association links cellular stress to impaired performance under the tested conditions, without isolating the energetic cost of antioxidant defense. California Current field comparisons also revealed taxon-specific oxidative responses in copepods and pteropods [62]. Such taxon-specific responses can reflect exposure history, damage or compensation. Their nutritional significance is clearest when antioxidant measurements are paired with intake, expenditure and life history endpoints.

Respiration, biochemical composition and gene expression resolve different aspects of compensation. In 4-day-old *Calanus hyperboreus* nauplii, combined warming and low pH increased oxygen uptake, whereas lipid and fatty acid (FA) composition remained statistically unchanged under the same treatments [63]. Maintained lipid composition therefore did not exclude a metabolic response. In *Limacina helicina antarctica*, combined stress affected survival and gene expression, with a stronger transcriptomic response to temperature in a 21-day experiment [64]. These joint stressor results require attention to the contributions of each treatment. Similarly, reduced copepod metabolism during acute freshwater exposure cannot automatically be interpreted as energy conservation [36].

Together, this framework relates acid–base regulation to metabolic adjustment while showing where evidence for nutritional costs remains incomplete. The evidence reviewed here falls into three levels: regulatory mechanisms established mainly in larger crustaceans; physiological responses measured in zooplankton, such as changes in respiration, metabolites or antioxidant activity; and energetic costs quantified directly as reduced scope for growth, which remain rare [65,66]. Figure 2 identifies the regulatory pathways, and Table 1 relates them to predictions for reserve depletion, growth and reproduction. Evaluating these predictions requires matched measurements of physiological function, resource expenditure and organismal performance over the same exposure interval. This approach distinguishes effective compensation from compensation that reduces resources available for production. It also provides the physiological basis for assessing how food availability and biochemical quality modify the response.

Table 1. Mechanistic pathways linking environmental acidification to zooplankton nutritional performance. Pathway-specific predictions distinguish direct physiological costs; evidence is taxon- and context-dependent. Mechanistic bases for the physiological pathways are derived partly from larger crustaceans, and nutritional predictions remain hypotheses.

Pathway	Mechanistic Basis	Nutritional Prediction	Diagnostic Evidence
Ion and acid–base regulation	Epithelial transporters maintain Na^+, Cl^-, H^+ and HCO_3^- gradients; buffering and excretion stabilize extracellular and intracellular pH.	Higher ATP demand can reduce energy for growth and reproduction; ionic failure can reduce activity and feeding.	ATPase activity, ion fluxes, hemolymph or intracellular pH, respiration and reserves with life history endpoints [43,46,47].
Molting and mineral balance	Ca^{2+} transport and bicarbonate delivery are coordinated with cuticle resorption and deposition.	Longer intermolt periods, incomplete mineralization or greater postmolt costs can delay development.	Molt stage, Ca^{2+} balance, bicarbonate transport, cuticle properties and growth [55,56].
Respiration and cellular metabolism	Acid–base compensation and altered mitochondrial metabolism change maintenance expenditure.	Reduced scope for growth, accelerated reserve depletion or delayed reproduction.	Joint measurements of oxygen consumption, ingestion, ATP, metabolites, lipid stores and production [60,65,66].
Oxidative defense	Reactive oxygen species (ROS) and antioxidant systems respond to low pH and co-stressors.	Resource diversion can limit tissue production and reproduction.	ROS, antioxidant enzymes, damage markers, growth and fecundity in the same cohorts [61].
Feeding behavior	Swimming, encounter rate, prey selection and clearance can change independently of prey concentration.	Lower ingestion or altered prey choice can reduce assimilated intake; unchanged ingestion can accompany poorer food quality.	Activity, clearance, ingestion and prey composition under matched exposure histories [21,67].
Prey biochemical quality	Acidification and nutrients alter prey stoichiometry, FA, sterols and amino acids.	Abundant carbon can remain nutritionally inadequate, reducing recruitment and transfer efficiency.	Reciprocal prey–consumer exposure with prey and consumer composition, recruitment and carbon transfer [68–70].

Table 1. *Cont.*

Pathway	Mechanistic Basis	Nutritional Prediction	Diagnostic Evidence
Assimilation and lipid conversion	Consumers differ in capacity to assimilate or biosynthesize essential compounds.	Stress can limit dietary compensation and reduce prey quality for predators.	Compound-specific tracers, consumer and egg lipids, and biosynthesis gene expression [71,72].
Cross-generational allocation	Parental condition, epigenetic responses and selection redistribute costs across generations.	Maintained adult performance can mask reduced offspring quality, delayed development or lower population growth.	Reciprocal parental–offspring exposures, reserves, egg quality, hatching, juvenile survival and common garden tests [73,74].
Community replacement	Differential sensitivity changes grazer size structure, feeding guilds and particle pathways.	Stable total abundance can conceal changes in prey value, grazing control or export.	Taxonomic and size structure, biochemical prey value, production and particle export [11,75].

4. Food-Mediated Effects on Zooplankton Nutrition

Acidification can alter food supply by changing prey abundance, community composition, and nutrient content, while the efficiency of resource conversion by consumers determines how much of the ingested material contributes to production. Food-mediated effects therefore link exposure chemistry to the resource available for growth and reproduction. Table 2 summarizes the evidence along this pathway, distinguishing changes measured in prey from those associated with consumer responses.

Table 2. Biochemical pathways linking acidification to zooplankton nutrition.

Pathway	Exposure Design	Biochemical Evidence	Trophic Response	Inference and Boundary
Algal FA [76]	*Thalassiosira pseudonana* → *Acartia tonsa*; crossed prey × consumer CO_2, nominal 380/740 µatm pCO_2. Algal realized pCO_2: 365 ± 120/915 ± 270 µatm.	Algal total FA: 28.1 → 17.4 fg cell^{-1}; lower PUFA:SFA and essential FA availability.	Median egg production: 34 → 5 eggs female^{-1} d^{-1} (low/low versus high/high treatments); development delayed.	Paired biochemical and reproductive measurements; rapid algal FA remodeling after transfer confounds separation of dietary and direct effects.
Excess dietary carbon [77]	*Rhodomonas salina* at 200/400/800 µatm pCO_2 → *Acartia tonsa*; juvenile and adult carbon processing compared.	CO_2-conditioned algal stoichiometry and surplus dietary carbon; consumer respiration and DOC release.	Higher algal CO_2 slowed copepodite development; surplus carbon was released through respiration in juveniles and as DOC in adults.	Qualitative support for stage-specific handling of altered prey stoichiometry; algal C:N:P changes and a fully crossed consumer exposure design were not verified.
Biochemical allocation [68]	*Acartia tonsa* with three algal species; prey × consumer factorial, nominal 400/1000 µatm pCO_2, one life cycle.	FTIR lipid:protein:carbohydrate ratios, C:N, ingestion, respiration and reproductive carbon budgets.	Dietary exposure reduced recruitment by ~30%; combined exposure reduced gross growth efficiency by 78% relative to control.	Factorial design separates exposure routes and links composition to carbon allocation. EPA/DHA-specific flux and fish transfer unmeasured.

Table 2. *Cont.*

Pathway	Exposure Design	Biochemical Evidence	Trophic Response	Inference and Boundary
FA supply × nitrogen [69]	Mixed phytoplankton → *Acartia tonsa*; 400/1000 ppm CO_2, four-day feeding; separate N-limited and N-depleted experiments with matched prey–consumer CO_2.	High CO_2 lowered dietary essential FA and PUFA concentrations; N depletion increased total FA and modified responses.	Median egg production fell 48–52% at high CO_2; hatching and naupliar survival also declined, with weaker effects on FA-rich N-depleted food.	Dietary chemistry and reproduction measured under matched exposure; no crossed prey–consumer treatments. Separate nitrogen experiments limit isolation of dietary and nitrogen effects.
Community FA transfer [78]	Raunefjord community → *Calanus finmarchicus* C5; mesocosm pCO_2 targets 280–3000 µatm, ~six weeks.	Nano- and picoplankton relative PUFA declined; copepod FA profiles resembled the nanoplankton fraction.	Copepod relative PUFA declined with CO_2 and over time at ≥1120 µatm; lifetime reproduction unmeasured.	Community biochemical changes extend to a primary consumer; relative FA profiles do not quantify absolute nutrient flux; prey size and availability covary and direct consumer effects are not isolated.
Maternal FA transfer [71]	Baltic mesocosm community → *Eurytemora affinis*; four weekly feeding assays along a CO_2 gradient.	Female PUFA concentrations were reflected in eggs and positively associated with offspring production; no pH effect on ORAC.	No detected pH effect on offspring production; production was positively associated with food and dinoflagellate biomass.	Female egg nutritional coupling with null acidification results; associations do not establish acidification-induced FA limitation or universal tolerance.
Community buffering [79]	Kiel Fjord phytoplankton with *Acartia tonsa*; pCO_2 targets 380–4000 µatm; source community pCO_2 ~1600 µatm.	Community composition and elemental quality; minor stoichiometric changes mainly at the highest CO_2.	Copepod abundance and egg production were similar across treatments.	Context-dependent buffering; FA unmeasured, most treatments below source community CO_2, copepods from stock culture.
Consumer compensation [72]	Benthic *Platychelipus littoralis*; factorial LC-PUFA-deficient diet, +3 °C warming and −0.4 pH units.	FA profiles and DHA-related Δ4 desaturase/elovl1a transcripts; dietary deficiency stimulated biosynthetic expression, but acidification did not increase it above baseline.	Relative DHA was maintained on the deficient diet at ambient temperature but declined with warming.	Consumer capacity can modify dietary vulnerability; transcripts indicate biosynthetic potential, not synthesis flux. This benthic species cannot establish a shared response in planktonic copepods.

Table 2. *Cont.*

Pathway	Exposure Design	Biochemical Evidence	Trophic Response	Inference and Boundary
Prey response × warming [70]	Three *Skeletonema marinoi* strains; 7/19 °C × 400/1000 ppm CO_2, 40-week conditioning; no consumer feeding assay.	FA composition, including n-3:n-6. Acidification reduced this ratio; warming alone and with acidification increased it.	Consumer uptake, reproduction and trophic transfer unmeasured.	Potential prey quality pathway and thermal interaction; FA ratios alone do not establish changes in absolute essential FA supply.
Phenolic accumulation [80]	Phytoplankton–zooplankton microcosms and mesocosms; nominal 395/1000 µatm pCO_2, matched trophic-level exposure.	Phytoplankton phenolics increased by 46–212%; zooplankton phenolics by 27.8% in mesocosms and 47.5% in microcosms.	Phenolic accumulation measured in copepod-dominated zooplankton; reproductive and nutritional effects untested.	Food-associated defensive compounds; no diet-only separation or fish feeding test. Accumulation does not establish zooplankton fitness loss or fish toxicity.

Notes. In Tables 2–4, exposure conditions retain the metrics and units reported in the original studies: pH (on the reported scale), seawater pCO_2 or fCO_2 (µatm) and gas-phase CO_2 (ppm). These metrics are not directly interchangeable, and values are not intended for direct comparison among studies. DHA, docosahexaenoic acid; DOC, dissolved organic carbon; EPA, eicosapentaenoic acid; FA, fatty acids; FTIR, Fourier transform infrared spectroscopy; LC-PUFA, long-chain PUFA; ORAC, oxygen radical absorbance capacity; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.

4.1. Prey Availability and Nutritional Quality

Prey availability depends on how primary production and community succession respond to chemical and nutrient conditions. CO_2 enrichment stimulated phytoplankton growth and altered community composition in nutrient-enriched freshwater mesocosms [81]. In a marine winter-to-summer succession experiment, increased biomass in some copepod size classes was attributed to indirect food web effects [82], whereas a low-nutrient Mediterranean experiment detected no significant CO_2 effects on feeding rates or egg, and naupliar standing stocks [83]. A nutrient-enriched coastal assemblage also maintained *Acartia tonsa* abundance and egg production across treatments [79]. The high CO_2 levels in the source water and variable exposure chemistry limit mechanistic attribution, but the contrasting results show that prey–consumer culture responses must be interpreted in the context of their food environment.

Changes in the prey field can affect nutrition even when consumers retain their capacity to gather food. During Little Rock Lake acidification, food availability and pH contributed to the decline in *Keratella cochlearis*, while the increase in *Keratella taurocephala* was not explained by those factors alone [84]. Maintained food gathering was reported in several dominant taxa despite changes in algal composition and selective feeding [67]. Microzooplankton grazing can further reshape the phytoplankton available to larger grazers [85]. Most copepods are omnivorous and feed on micro-sized particles, including phytoplankton, detritus and microzooplankton such as protists and rotifers [86,87]. Many species select prey according to size, motility and nutritional quality, and ciliates and other heterotrophic protists can contribute substantially to their diets [87,88]. Thus, phytoplankton biomass alone does not indicate the amount of prey available to a particular consumer.

Nutritional quality introduces a second constraint: additional dietary carbon may not be accompanied by sufficient phosphorus or essential FA [18]. In freshwater experiments, CO_2 enrichment stimulated algal growth but lowered the algal P:C ratio and reduced *Daphnia* growth, and phosphate enrichment of the high-CO_2 algae improved consumer

growth, supporting a dietary phosphorus mechanism [89]. A later *D. magna* feeding study found increased algal biomass together with lower P:C ratio and omega-3 polyunsaturated fatty acid (PUFA):C ratios, consumer growth declined on green algal and mixed diets, and responses to warming depended on the diet [90]. These results link carbon enrichment to nutritional imbalance under the tested nutrient and dietary conditions.

Experiments that separate prey and consumer exposure provide the clearest test of the dietary contribution. In a crossed *A. tonsa* experiment, prey-only exposure reduced naupliar recruitment by approximately 30%; combined exposure maintained female carbon ingestion but reduced gross growth efficiency, calculated as egg carbon production relative to ingestion, by 78% [68]. This shows a lower fraction of ingested carbon allocated to reproductive production. Paired reductions in algal FA content and copepod egg production are also consistent with a nutritional pathway, although rapid lipid remodeling after prey transfer complicated its separation from direct exposure [76]. Mixed-diet experiments likewise found reduced reproduction under matched high-CO_2 prey and consumer exposures. Differences in offspring responses between nitrogen contexts also involved prey composition and collection date, preventing attribution to nitrogen status alone [69].

Biochemical supply also changes through species turnover and responses within individual prey species. In prolonged cultures, *Skeletonema marinoi* strains showed different FA responses to warming and acidification [70]. In natural mesocosms, changes in plankton size composition and lower relative PUFA in the nano-sized fraction accompanied lower relative PUFA in *Calanus finmarchicus* [78]. These relative profiles indicate potential dietary changes, but absolute FA supply also depends on prey biomass and ingestion. Phytoplankton and zooplankton phenolic contents increased under matched acidification treatments, extending the range of possible biochemical effects; reproductive consequences and an isolated dietary route were not established [80]. Collectively, these studies support context-dependent changes in prey quality, rather than a uniform nutritional deterioration across communities. These contrasting outcomes can be attributed mainly to two factors. First, nutrient status modifies whether CO_2-stimulated carbon production dilutes phosphorus and essential FA in prey [69,89,90]. Second, food quantity and diversity can buffer quality deficits, so that consumer performance may be maintained [71,79]. Prey-only and community studies indicate potential dietary changes, whereas crossed prey–consumer experiments provide the strongest evidence for a dietary pathway [68,76].

4.2. Resource Conversion and Nutritional Compensation

After ingestion, elemental imbalance can alter the fate of dietary carbon. When *A. tonsa* consumers remained at ambient CO_2, feeding carbon-enriched *Rhodomonas salina* slowed development despite abundant food [77]. Juveniles released more carbon through respiration than as DOC, whereas adults showed the opposite balance: adult DOC release was not a monotonic response to prey CO_2 treatment. These measurements show that carbon handling differs between developmental stages and that maintained ingestion can coexist with reduced production. Carbon release and development were quantified, whereas assimilation efficiency was not measured. Consumer biochemical composition can also be modified by nutrient retention and biosynthetic regulation. The benthic copepod *Platychelipus littoralis* maintained relative docosahexaenoic acid (DHA) on a long-chain-PUFA-deficient diet at ambient temperature, whereas warming reduced absolute DHA content and acidification constrained induction of selected biosynthesis-related genes [72]. Deficient food was supplied independently of acidification, allowing consumer regulation under poor nutrition to be examined. The results suggest conditional nutritional compensation, but gene expression does not quantify newly synthesized PUFAs, and this benthic response cannot establish the biosynthetic capacity of planktonic copepods.

Maternal allocation connects consumer nutrient status to the next generation. In Baltic mesocosms, *Eurytemora affinis* maternal PUFA content covaried with egg PUFA and naupliar production, although no significant pH effect on offspring production was detected over the tested interval [71]. Together with stage-dependent carbon utilization, these associations indicate that prey composition alone cannot predict the nutrients retained by consumers or transferred to their offspring. Accordingly, maintained biochemical composition or reproductive output indicates maintained performance, but does not by itself demonstrate successful nutritional compensation or unchanged resource allocation, because these traits may be preserved at the expense of reserves, development time or offspring quality. Food-mediated effects are therefore best resolved by linking prey availability and nutrient content to food intake, resource conversion, and subsequent production. These connections determine whether the tested food conditions offset or reinforce physiological demands and provide the nutritional basis for interpreting growth, reproductive allocation and population responses.

5. Growth, Reproduction and Population Performance

Physiological demands and nutritional constraints jointly influence growth, reproduction, and survival. Table 3 summarizes these interrelated responses under specific exposure and feeding conditions. However, growth among survivors does not account for losses from the original cohort. In food-supplied *Eodiaptomus japonicus*, nitric acid exposure reduced survival to adulthood despite similar estimated somatic growth, whereas reproductive responses were nonmonotonic [91]. Similarly, a freshwater CO_2 experiment conducted at pH 6.7, representing an extreme freshwater acidification scenario, found no change in the adult growth rate of *Daphnia magna*, despite slower juvenile growth, delayed maturation, reduced survival, and smaller offspring [92].

Table 3. Evidence linking acidification to zooplankton energy allocation and life history performance. Values apply to the stated exposure and food conditions and are not universal thresholds.

Design	Taxon, Exposure and Food Context	Reported Response	Inference Boundary
Arctic populations [66]	*Calanus glacialis* C4/C5 from two Svalbard populations and Disko Bay; pH response norms.	At pH 7.87, C4 scope for growth fell 19% in Kongsfjord and 50% in Billefjord; Disko Bay C4 and all C5 groups showed no comparable effect.	Population and stage differences preclude a single species-wide threshold.
Two generations [65]	*Calanus finmarchicus*, limited food; ~380, 1080, 2080 and 3080 µatm pCO_2.	Parental development was delayed at 2080 µatm, without a detected F1 delay; high pCO_2 increased maintenance constraints.	Limited to food restriction and extreme scenarios.
24 h freshwater exposure [36]	*Daphnia pulicaria*, *Eodiaptomus japonicus* and cyclopoids; pH 4.0–8.0 at 10 and 20 °C.	Mortality >50% below pH 4.6 for *Daphnia*/cyclopoids and below 5.6 for *E. japonicus*; copepod metabolism declined at pH 4.6–7.0.	Acute thresholds indicate taxon sensitivity, not chronic population performance.
Freshwater life table [91]	*Eodiaptomus japonicus*, pH 7.8/6.8/6.0, sufficient food.	Survival to adulthood <20% under acid stress; estimated population growth fell 2.9% per 0.1-unit pH decrease.	Relatively stable somatic growth among survivors reveals endpoint trade-offs.

Table 3. *Cont.*

Design	Taxon, Exposure and Food Context	Reported Response	Inference Boundary
53-day overwintering [93]	*Calanus glacialis*, 0/4 °C × pH ~8.0/7.4–7.3 during polar night.	Combined warming–acidification doubled mortality; warming accelerated molting, with lipid exhaustion projected 1–4 months before the spring bloom.	Seasonal reserve dynamics reveal delayed risks that active feeding assays would miss.
Chronic freshwater physiology [94]	Acclimated *Daphnia pulex* cultures, pH 7.8/6.0/5.5.	Hemolymph bicarbonate fell 30–65%; compensation involved increased circulation, ventilation and metabolism, with reserve and reproductive costs.	Compensation is not cost-free; responses depend on species and medium.
21-day life table [95]	*Daphnia pulex*; sulfuric acid addition to pond water; paired acute and chronic exposures.	At pH ≤ 5.0, mortality and delayed maturity reduced population growth potential; survivors maintained brood size.	Acute lethality, survivor fecundity and population growth are distinct endpoints.
Short-term vital rates [41]	*Acartia grani* and *Oithona davisae*; pH ~8.18 versus 7.78, four-day preconditioning, characterized prey.	No clear feeding, respiratory or reproductive effects; prey FA profiles unchanged.	Endpoint-specific tolerance; delayed or food-dependent effects remain possible.
Lifetime reproduction [96]	*Tigriopus japonicus*; 56 days, pH 8.0/7.7/7.3/6.5.	Survival unaffected; development, hatching or lifetime offspring output changed in specific low-pH treatments.	Dose- and endpoint-specific responses; pH 6.5 is extreme, not a general end-of-century mean.

Resource balance provides a mechanistic link between these endpoints, but evidence beyond body size is needed to assess the proposed costs of resource allocation. Under food-limited conditions, *Calanus finmarchicus* exhibited higher respiration rates and a lower estimated scope for growth, without increased feeding among the experimental CO_2 gradient [65]. In *Daphnia pulex* acclimated to low pH, a reduction in visible energy reserves was associated with smaller brood size at pH 5.5 [94]. These observations connect physiological demand with condition and production under the tested regimes. In *Tisbe battagliai*, reduced body length at pH 7.82 and 7.67 was associated with nonmonotonic responses in naupliar production among three analyzed generations [97]. Such concurrent changes are consistent with altered resource allocation, although the experiment did not quantify the energy budget.

Adult reproductive output represents the first step in offspring production, and its persistence must be assessed over a defined exposure period. Short exposures to the projected end-of-century CO_2 conditions produced no significant effects on egg production or hatching in two calanoid copepods, *Centropages typicus* and *Temora longicornis* [98]. Similarly, *Acartia grani* and *Oithona davisae* showed no significant changes in reproductive performance after short-term preconditioning at pH 8.18 and 7.78 [41]. Conversely, rapid pH reduction increased egg production in *A. bifilosa* and improved its early developmental index at 30 h, whereas hatching success remained unchanged and the developmental difference disappeared by 66 h [99]. These contrasting responses indicate that short-term egg production may remain stable or increase without necessarily indicating improved subsequent recruitment.

Following eggs and juveniles reveals constraints that adult reproductive output alone may not capture. *Calanus glacialis* maintained egg production, but hatching success was

reduced during the observation period at an extremely low pH of 6.9, consistent with delayed hatching [100]. Baltic *Acartia* mesocosms produced smaller females, while egg transfer experiments revealed lower relative hatching success and developmental rates in high-CO_2 maternal water than in ambient Baltic water [101]. The transfer results suggest incomplete parental buffering under those conditions. In acute *A. tonsa* assays, a nominal pCO_2 of 1000 µatm increased naupliar mortality and reduced recruitment estimated from egg production and hatching success [102]. That recruitment metric excluded subsequent naupliar survival; consequently, the two effects indicate distinct potential bottlenecks rather than a directly measured intrinsic population growth rate.

Longer observation periods allow assessment of whether these responses persist across reproductive cycles and exposure histories. In a 56-day *Tigriopus japonicus* experiment, survival remained unchanged despite delayed naupliar development and reduced egg sac viability at pH 6.5. Cumulative offspring production was lower at pH 7.3, indicating that different endpoints did not exhibit a consistent monotonic response to pH [96]. Five-generation experiments further demonstrated that constant and progressively declining pH conditions resulted in distinct developmental and reproductive responses [103]. Within the declining pH sequence, increased ingestion rates and ATPase activities were associated with reduced offspring production; however, these physiological measures were not compared between exposure modes [103].

Demographic interpretation requires integrating age-specific survival with the number and timing of offspring. A 21-day *D. pulex* life table study showed that lower survival and delayed first reproduction reduced population growth potential at low pH. Brood sizes remained broadly comparable across several treatments among females that survived to reproduce [95]. The *E. japonicus* life table study likewise estimated lower potential population growth under acid stress despite unchanged somatic growth [91]. These estimates demonstrate that fecundity among survivor or adult size can provide a different picture of population performance than cohort-level measures. They reflect laboratory growth potential under specific feeding and water chemistry conditions, whereas field abundance also depends on ecological interactions and dispersal. At present, population consequences are therefore estimated mainly from individual life history data, from which population growth rates can be calculated. Direct demographic measurements under acidification remain scarce. Whole-lake acidification experiments provide direct tests in freshwaters. At Little Rock Lake, laboratory bioassays were consistent with the decline in some rotifer populations, whereas other population changes could not be explained by pH and food alone [84]. Comparable ecosystem-scale manipulations are not feasible in the ocean, where mesocosm experiments provide the closest community-level tests [78,101].

Other experimental and field endpoints provide complementary evidence at different scales. Four coastal copepods (*Acartia pacifica*, *Acartia spinicauda*, *Calanus sinicus* and *Centropages tenuiremis*) exhibited species-specific survival and reproductive responses across CO_2 concentrations of 800–10,000 ppm, with no significant effect on survival or egg production at 800 ppm [104]. Adverse effects began at 2000 ppm for survival, egg production, and hatching success in *C. tenuiremis* and for survival and hatching success in *A. spinicauda*. In contrast, egg production of *A. spinicauda* and *C. sinicus* declined significantly only at 5000 and 10,000 ppm, respectively, and *A. pacifica* was not significantly affected [104]. Differences in diets and temperatures complicate interspecific comparisons, and the highest treatments represent extreme exposures. *Pseudodiaptomus serricaudatus* exhibited increased mortality among adult males and changes in final culture abundance and stage composition in separate assays; however, stage ratios did not directly quantify development time or intrinsic population growth rates [105]. Acute sensitivity of *D. galeata mendotae* neonates to acidification was consistent with its reduced occurrence and biomass in acidic Ontario lakes [106].

These findings link individual performance to population vulnerability while maintaining distinctions among acute tolerance, chronic production, and long-term persistence in natural environments.

6. Trophic Transfer and Ecosystem Functions

These individual responses influence food web function through changes in zooplankton production, prey characteristics, and community composition. For predators, food supply depends on the quantity, size and biochemical composition of prey they can capture. In a factorial mesocosm experiment, warming and elevated CO_2 induced stage-specific changes in copepod abundance and body size, as well as changes in specific FA ratios in adult *Paracalanus* [107]. These measurements indicate potential changes in prey quantity and quality, but neither fish feeding nor larval performance was directly assessed. Abundance alone therefore cannot determine whether the nutritional supply available to higher trophic levels has changed.

Community replacement represents a broader pathway through which individual sensitivity can alter prey availability. Surveys of acidified freshwater lakes have documented reduced zooplankton richness, shifts in the relative abundance of cladocerans and copepods, and the dominance of acid-tolerant taxa in some rotifer communities [10,108–110]. These patterns reflect freshwater acidity gradients and associated chemical conditions; their applicability to marine CO_2 enrichment requires separate evaluation. Changes in taxonomic composition may alter grazing patterns and the size range of prey available to fish larvae, which depend largely on zooplankton as food, although the nutritional consequences have not been quantified. The loss of predators or changes in predation pressure can also contribute to shifts in community composition, complicating the attribution of these changes to direct zooplankton sensitivity [108]. Interactions between predators and acidification can further modify zooplankton availability to other consumers. In a 10-day microcosm experiment, elevated CO_2 and the presence of the cubozoan *Carybdea rastoni* reduced calanoid abundance more than expected based on their independent effects [111]. The experiment showed stronger predator-dependent depletion under the tested conditions but did not distinguish increased prey vulnerability from increased predator feeding activity. Such community-level outcomes cannot be predicted from zooplankton tolerance alone because actual abundance also depends on food web interactions.

Evidence from fish-containing food webs is needed to determine whether changes in zooplankton availability affect the performance of higher-level consumers. The *A. tonsa* experiment measured carbon transfer from phytoplankton to zooplankton, rather than to fish [68]. In contrast, a large-scale mesocosm experiment reported higher survival of Atlantic herring larval under elevated CO_2, which was associated with greater availability of plankton food [112]. This result supports an indirect food web benefit under the tested conditions and highlights the need to avoid assuming that acidification uniformly reduces food availability for fish. Larval survival, however, does not quantify biochemical assimilation or trophic transfer efficiency. Interpreting the responses of higher-level consumers consequently requires matching prey availability and composition to predator feeding, growth, and survival over the same exposure period. Direct evidence of altered trophic transfer under acidification is therefore currently limited mainly to the phytoplankton–zooplankton trophic link [68,76,78], whereas transfer from zooplankton to fish and other predators is supported mainly at the level of potential mechanisms. Direct tests at higher trophic levels remain difficult, because zooplankton must be cultured under acidification for long periods and in sufficient quantities to meet the energy demands of higher-level consumers.

Zooplankton also contribute to ecosystem functioning by respiring organic carbon and releasing nutrients into the dissolved pool. In a subtropical oligotrophic mesocosm

experiment, elevated CO_2 reduced biomass-specific respiration and ammonium excretion in mesozooplankton, while protein biomass showed no significant response to CO_2 enrichment [113]. After simulated upwelling, a harmful algal bloom under elevated CO_2 conditions coincided with reduced mesozooplankton biomass and altered metabolic responses [113]. These findings link acidification to metabolic processing, but biomass-specific rates cannot determine the direction of changes in community nutrient regeneration. Assessing this feedback requires measurements of both metabolic rates and zooplankton biomass, along with information on nutrient limitation and subsequent primary production.

Carbon can also enter sinking particle pathways, which are influenced by community composition and particle processing. In a warming–acidification mesocosm experiment, appendicularian abundance increased under warmer conditions, while elevated CO_2 alone did not increase their abundance [75]. The increase in appendicularians altered the grazer community and the size distribution of suspended particulate carbon. Discarded filtration houses and fecal pellets were suggested as potential export pathways, but sedimentation was not directly measured [75]. The experiment therefore suggests a possible redistribution of carbon rather than providing direct evidence of increased carbon export. Evaluating this proposed pathway requires direct measurements of sinking particles alongside the organisms involved in their production and processing.

Direct particle measurements also demonstrate that changes in suspended material cannot be simply extrapolated to carbon export. A companion study of the subtropical experiment directly measured sinking particle composition [114]. Post-bloom sediment trap material exhibited lower C:N and C:P ratios under elevated CO_2. Reduced preferential remineralization of nitrogen and phosphorus relative to carbon was suggested as a possible explanation for this pattern, but the mechanism was not experimentally confirmed. The enclosures with the highest CO_2 levels retained more particulate material after the bloom, although the regressions of sinking carbon flux against CO_2 were not statistically significant [114]. These measurements from shallow enclosures do not establish long-term deep ocean carbon sequestration. Overall, the reviewed studies address different components of carbon cycling. Community composition and suspended particles reflect particle production and processing, whereas direct measurements of sinking particles quantify short-term sinking flux. Long-term sequestration also depends on how deep particles sink before remineralization, which was not measured in these enclosure experiments. Assessing ecosystem consequences therefore requires integrated measurements of trophic supply, nutrient regeneration, and particle export, as these processes may respond differently to acidification.

7. Context Dependence, Adaptation and Recovery

7.1. Physiological and Environmental Modifiers of Acidification Responses

Warming, food availability and seasonal conditions can change the direction and magnitude of acidification responses because they act on different components of the resource budget. Food availability determines resource acquisition, warming raises maintenance costs and accelerates development, and seasonal dependence on stored reserves constrains allocation when feeding ceases. In the Humboldt upwelling system, a five-month study linked pH and food availability to ingestion, egg production, and egg traits in *A. tonsa* [115]. Egg production was similar among pH categories at higher chlorophyll concentrations, although egg production efficiency remained positively associated with pH. These observations suggest that food availability can moderate reproductive responses to acidification, while covariation in hydrographic conditions limits the attribution of these responses to pH alone. Seasonal dependence on stored energy reserves can reveal vulnerabilities that may be overlooked during active feeding. During the Arctic polar night, a 53-day exper-

iment involving unfed *C. glacialis* copepodites found that warming accelerated molting and reduced lipid fullness, while combined warming and severe acidification increased mortality [93]. Reserve depletion before the spring bloom was predicted based on observed lipid trajectories, rather than directly measured. Freshwater bioassays using sulfuric acid also revealed habitat-specific and seasonal differences in acute survival [116]. Thus, sensitivity depends on exposure timing and physiological history, as well as taxonomic identity. Warming can also modify acidification responses differently across traits. Adult *T. japonicus* exposed to brief heat shocks exhibited temperature-dependent feeding and respiration rates, with limited direct effects of CO_2 [117]. In an *A. tonsa* mesocosm experiment, increasing pCO_2 enhanced growth in terms of carbon at both temperatures, whereas mortality decreased at 13 °C and increased at 19 °C [118]. A separate factorial mesocosm experiment revealed antagonistic interactions between warming and acidification affecting naupliar and copepodite abundance [107]. Faster growth and improved survival therefore reflect distinct outcomes, and an antagonistic interaction does not necessarily restore abundance to control levels.

Metal interactions also depend on contaminant identity, concentration, and exposure history. Elevated CO_2 increased cadmium accumulation and acute toxicity in *T. japonicus* [119], but reduced nickel toxicity during multigenerational exposure, with lower nickel burdens observed in later generations [120]. In *Pseudodiaptomus annandalei*, elevated CO_2 reduced mercury accumulation and certain reproductive impairments [121]. Controlled seven-day experiments with *T. japonicus* further revealed reduced mercury accumulation under fluctuating CO_2 enrichment compared with steady CO_2 enrichment [122]. These studies link altered toxicity to body burden and potential physiological compensation; however, they do not establish a universal mechanism involving metal speciation or demonstrate that environmental variability generally mitigates acidification stress. Natural gradients illustrate how these context-dependent responses occur within complex environments. Near shallow hydrothermal vents, mesozooplankton mortality was associated with pH, temperature, and proximity to vents in waters that also contained sulfide [123]. Surveys in the Yellow Sea and Jiaozhou Bay likewise revealed associations between community attributes, temperature, and pH, with contrasting patterns of abundance and biomass in Jiaozhou Bay [124,125]. These spatial and seasonal observations highlight context-dependence responses, while their limited sampling periods and covarying environmental conditions constrain conclusions about long-term trends or causal interactions among stressors. Apparent resilience under these conditions therefore requires separate assessments of environmental buffering and inherited responses.

7.2. Parental Effects, Epigenetic Responses and Genetic Adaptation

Exposure history can alter performance among generations through parental condition, epigenetic regulation, or genetic selection. Table 4 summarizes the experimental designs and evidence underlying these mechanisms. F0–F2 comprises three generations. Continuous exposure cannot separate inherited parental effects from each generation's direct exposure. DNA methylation, histone modification and allele frequency change provide distinct evidence. Continuous exposure from F0 to F2 in *Calanipeda aquaedulcis* impaired reproductive performance, with severe losses at the lowest pH levels [126]. Such results indicate reproductive costs extending beyond the parental generation, but continued exposure of offspring makes it difficult to isolate parental effects. Improved performance over successive exposed generations consequently requires further mechanistic evidence before it can be attributed to inheritance or genetic adaptation.

Table 4. Responses among generations and adaptation to environmental acidification.

Organism	Exposure and Temporal Design	Performance Across Generations or Histories	Mechanistic or Discriminating Evidence	Interpretation and Boundary
Pseudo-calanus acuspes [73,127]	400/900/1550 µatm pCO_2, F0–F2; mature F2 underwent 21-day reciprocal CO_2 transplants.	At 900 versus 400 µatm, F2 fecundity fell 29% and metabolism rose ~10%; cross-generational effects partly buffered greater impairment at 1550 µatm.	Reciprocal transplant phenotypes plus allele frequency changes enriched in oxidative phosphorylation and ribosomal functions.	Persistent cross-generational responses and selection evidence; transplant phenotypes alone cannot identify inheritance mechanisms. Paired studies share one experiment.
Acartia sp. [101]	Baltic mesocosms, fCO_2 365–1231 µatm; egg transfers to new CO_2 conditions tested maternal buffering.	Smaller females at elevated fCO_2; maternal buffering of hatching and development appeared limited at high exposure.	Egg transplantation, female antioxidant capacity and reproductive endpoints; no allele frequency test.	Maternal/embryonic plasticity with possible limits; food covaried with treatment; no genetic adaptation claim. Same campaign as Almén et al. (2016) [71].
Brachionus koreanus [128]	Asexual rotifer, F0–F2; pH 8.0/7.7/7.3 and parental exposure lasting 1/3/7 days.	F0 growth, fecundity and lifespan declined; some effects diminished in F1/F2. DNA damage recovery depended on parental exposure duration.	Oxidative stress and DNA damage endpoints; histone modifications associated with DNA repair genes.	Cross-generational mitigation and chromatin-associated regulation. Nutritional flux unmeasured; pH 7.3 severe.
Paracyclop-ina nana [74]	pH 8.0/7.7/7.3; F0–F2 and later follow-up, with continued exposure and return-to-control branches.	Fecundity and female sex ratio recovered by F2 in exposed lineages.	Inbred lines, methylomes, transcriptomes and experimental DNA methylation manipulation.	Mechanistic support for epigenetic plasticity; reproductive recovery does not establish restoration of all nutritional functions or DNA sequence adaptation.
Tigriopus japonicus [103]	F0–F4; constant pH 8.1/7.7/7.3 versus descending 8.1 → 7.9 → 7.7 → 7.5 → 7.3.	Descending exposure increased ingestion but impaired development and reproductive allocation; constant exposure elicited weaker responses.	Ingestion, life history traits, ATPase activity and kinetics; no common garden or allele frequency test.	Trajectory-dependent compensation; increased ingestion does not establish energetic compensation. The rapid pH decline is experimental.
Calanipeda aquaedulcis [126]	Estuarine copepod, F0–F2 over 55 days; continuous pH 8.1/7.5/7.0/6.5.	Survival and reproduction remained impaired; beneficial parental responses did not fully offset acidification effects.	Multigenerational life history measurements under continuous exposure; no reciprocal return-to-control treatment.	Incomplete buffering; continuous exposure cannot separate inherited parental effects from direct exposure. pH 7.0/6.5 does not represent typical near-future marine means.
Acartia tonsa [129,130]	25 generations; replicated 18/22 °C × 400/2000 µatm pCO_2 factorial evolution.	Joint stress initially reduced fitness ~56%; reproduction recovered by generation 3, but fitness averaged ~9% below control.	Life histories and replicated allele frequency trajectories distinguish selection from drift and reveal interacting polygenic responses.	Rapid, incomplete genetic adaptation to joint stress; warming dominates parts of the genomic response. Recovery is not attributable to acidification alone; paired studies are not independent replicates.

Table 4. *Cont.*

Organism	Exposure and Temporal Design	Performance Across Generations or Histories	Mechanistic or Discriminating Evidence	Interpretation and Boundary
Acartia tonsa [131]	65 generations, 18 °C/400 µatm versus 22 °C/ 2000 µatm pCO_2; reciprocal transfers and ambient follow-up.	Adapted joint stress lineages had reduced thermal tolerance and altered thermal plasticity.	Thermal tolerance reaction norms after transfer; same *A. tonsa* evolution program as above.	Chronic joint stress adaptation may carry thermal extreme costs; acidification alone is not shown to lower heat tolerance. Nutritional transfer unmeasured.
Leptodiaptomus minutus [132]	Historically acidified Canadian lakes after 6–8 years of recovery; seven-day field transplants and 32-day acidic/ circumneutral laboratory assays.	Acid tolerance differed with exposure history and declined with longer chemical recovery; tolerant populations incurred fitness costs at circumneutral pH.	Population comparisons and transplants across current pH conditions; no genomic measurement.	Phenotypes are consistent with context-dependent tolerance and adaptive reversal; historical population differences and maternal effects remain possible.
Leptodiaptomus minutus [133]	Resting eggs from two historically acidified lakes, ~100 years/~200 generations; resurrected F1 lifetime assays at pH 4.5/6.5.	Historical acidification cohorts had greater tolerance; recovery cohorts lost it. Survival, fecundity and development varied among cohorts.	Resurrection ecology, temporal whole-genome data and demographic models consistent with selection and population rescue.	Historical genomic support for evolutionary rescue; F1-only assays did not standardize maternal effects, and hatching methods differed by cohort.

Epigenetic studies provide one possible explanation for buffering of responses in later generations. In *Paracyclopina nana*, analyses of the methylome and transcriptome, environmental transfer experiments, and methylation manipulation linked reproductive recovery to DNA methylation plasticity [74]. In *Brachionus koreanus*, the mitigation of adverse effects was associated with DNA damage responses and histone-related regulation [128]. A recent *T. japonicus* study linked F4 acclimatization to fluctuating high-CO_2 and cadmium exposure with DNA hypomethylation and changes in gene expression [134]. These studies provide evidence of distinct regulatory responses: experimental manipulation provides stronger causal evidence than molecular associations alone, but neither approach demonstrates DNA sequence evolution. Evidence for genetic adaptation requires more than phenotypic differences among habitats. Natural *A. tonsa* populations exhibited genetic and phenotypic differentiation but showed no consistent population-specific reproductive responses to low pH in short-term acclimation experiments [135]. In *Pseudocalanus acuspes*, reciprocal transfer experiments after F0–F2 exposure distinguished reversible plasticity at 900 µatm from transgenerational buffering at 1550 µatm [127]. Fecundity remained approximately 29% below control levels in continuously exposed lineages. Genomic analyses of the same populations identified evidence of selection involving oxidative phosphorylation and ribosomal functions [73]. These two studies provide complementary evidence, linking incomplete physiological buffering to potential genetic responses.

Experimental evolution further demonstrates that rapid adaptation can involve persistent fitness costs. In a 25-generation experiment, combined warming (22 °C) and acidification (2000 µatm pCO_2) initially reduced *A. tonsa* fitness by approximately 56% through lower egg production and hatching success. However, both traits recovered by the third generation, and fitness thereafter remained, on average, approximately 9% below control levels, consistent with rapid evolutionary adaptation [129]. Survival then declined af-

ter generation 15, as antagonistic interactions between warming and acidification limited full evolutionary rescue. In contrast, acidification alone did not act as a strong selective force [129]. These results suggest that predictions based on first-generation responses may overestimate long-term fitness losses, although adaptation may be incomplete and its extent under food limitation remains uncertain. Genomic analyses from the same experiment provided evidence of selection acting on standing genetic variation and interactive responses to the two stressors [130]. Lineages maintained through generation 65 exhibited reduced acute thermal tolerance, although their performance at high temperature improved after nine generations under ambient conditions [131]. Adaptation to chronic combined stress can therefore increase vulnerability to thermal extremes; however, its benefits and costs cannot be attributed to acidification alone. Freshwater records provide a historical context for these responses to acidification and subsequent recovery. In *Leptodiaptomus minutus*, acid tolerance declined over time following the recovery of lake pH, and acid-tolerant adults exhibited reduced survival at circumneutral pH [132]. Resurrection ecology combined archived resting eggs, temporal genomics, and demographic reconstruction to provide evidence for probable evolutionary rescue and subsequent adaptive reversal [133]. Maternal effects and hatching history continued to influence F1 trait comparisons, limiting the ability to distinguish their respective contributions to individual phenotypes. Taken together, acclimation, developmental plasticity, parental effects, epigenetic regulation and genetic adaptation differ in their mechanisms and evidential requirements and should not be treated as equivalent forms of buffering (Table 4). Nutritional endpoints were rarely measured across generations, so whether these processes restore resource acquisition and allocation, and thereby long-term ecological performance, remains to be tested.

7.3. Community Recovery After Chemical Improvement

At the community scale, improvements in water chemistry can facilitate recovery but do not eliminate all habitat constraints. Emission controls have reduced acid deposition and improved lake water chemistry in many regions [12]. Historical monitoring in Ontario (Canada), nevertheless, showed that declining sulfate concentrations did not consistently lead to increases in pH or alkalinity, while drought-related sulfate mobilization and reduced base cation export partly offset the effects of lower atmospheric inputs [136]. Biological recovery therefore depends on the resulting environmental conditions, the availability of colonists, and their successful establishment [137]. This process is influenced by both changes in environmental filters and the exposure histories of populations capable of recolonization.

Residual ionic constraints can influence which grazer assemblages re-establish. A factorial in-lake experiment revealed higher abundances of several small, acid-tolerant cladocerans such as *Daphnia catawba* and *Daphnia ambigua* under low-calcium conditions [11]. Long-term monitoring in Norway documented increases in crustacean richness and functional richness based on traits during 1997–2020, with recovery trajectories varying among lake types characterized by different calcium concentrations and humic conditions [138]. Clear-water lakes exhibited increased functional richness, whereas very-low-calcium humic lakes showed little biological change and no significant increase in pH. Such differences highlight the importance of interpreting recovery in the context of lake type, rather than assuming a uniform biological response to regional reductions in acid deposition.

Successful re-establishment also depends on recruitment and food web interactions. Surveys in southern Norway suggest species-specific contributions from resting eggs, surviving populations, and external dispersal [139,140]. Following the liming of Lake Gårdsjön, fish introduction reduced the abundance of invertebrate planktivores and was followed by increased cladoceran abundance and reduced rotifer abundance [141]. Predator restoration

can thus indirectly reshape recovering communities. Species richness and trait-based functional richness reflect restored ecological potential but do not quantify grazing, production, or biochemical prey quality [138]. Species recovery and the restoration of nutritional support for predators are therefore related outcomes, but the extent to which they correspond remains unclear.

8. Evidence Gaps and Research Priorities

Establishing a link between acidification exposure and the quantity and nutritional quality of zooplankton production remains a major evidence gap. Freshwater studies more often document long-term community trajectories, whereas marine experiments more often quantify carbonate chemistry and biochemical traits. Comparisons between experimental sensitivity and natural assemblages further highlight the need to distinguish individual responses from patterns of abundance in the field [142]. Table 5 summarizes the measurements and experimental designs needed to connect these scales. Its rows follow the causal chain, and the first links represent the most fundamental gaps. Consistent exposure characterization and matched measurements of intake, expenditure and production are required before population and food web consequences can be interpreted. Multigenerational, field recovery and food web studies address more advanced questions that build on these measurements, and their specific designs depend on the species, habitat and life history of the study organism. Meaningful comparisons of biological responses first require consistent characterization of the actual exposure conditions. Marine studies should report carbonate system parameters and the pH scale used, alongside temperature, salinity, dissolved oxygen, and temporal variability. Freshwater studies should additionally identify the acidification pathway and measure ANC, relevant ions and metals, DOC, and hydrological conditions. Water chemistry should be monitored throughout the biological observation period, with measurements distinguishing contemporary conditions, episodic extremes, and future scenarios. Modeled pH values at plankton surfaces may differ from those in bulk water [143].

Within a defined chemical environment, crossed prey–consumer exposure designs can distinguish direct physiological effects from food-mediated effects. Food quantity should also be varied when resource limitation is relevant. Prey biomass, size composition, and biochemical content should be measured under the growth conditions and following transfer to the feeding environment. Repeated measurements during the period when prey are available to consumers would capture biochemical remodeling that may complicate the attribution of dietary effects. These designs should determine whether changes in prey abundance or nutritional quality precede changes in intake and production, rather than inferring a dietary mechanism from consumer composition alone. Distinguishing these pathways also requires measurements of resource conversion and energy expenditure over comparable time intervals. Clearance and ingestion should be measured alongside egestion, assimilation, or tissue incorporation, while respiration and activity should be assessed together with energy reserves and production. Stable isotope or compound-specific tracing can help quantify the incorporation of dietary carbon and essential compounds. Measurements of absolute nutrient supply and, where feasible, biosynthetic fluxes should complement relative FA profiles and gene expression data. Integrating these measurements would help distinguish reduced resource acquisition from increased maintenance costs and identify compensatory responses that reduce the resources available for growth or reproduction.

Table 5. Minimum measurements and preferred designs for resolving the causal chain from acidification exposure to trophic transfer.

Causal Link	Minimum Reporting Set	Preferred Design	Inference to Avoid
Exposure	Marine: pH, pCO_2 or DIC, alkalinity, temperature, salinity, oxygen. Freshwater: temperature, pH, ANC, metals, DOC, hydrology.	Time-resolved chemistry spanning mean conditions and realistic extremes.	Treating pH alone as a comparable measure of acidification between marine and freshwater ecosystems.
Prey field	Biomass, taxonomic and size composition, C:N:P, essential FA, sterols and amino acids.	Cross prey growth and consumer exposure chemistry; characterize prey before and after feeding.	Inferring food quality from chlorophyll or total carbon alone.
Acquisition and assimilation	Swimming or encounter rate, clearance, ingestion, egestion, assimilation and compound incorporation.	Pair feeding rates with assimilation or egg composition; use stable isotope or compound-specific tracing when feasible.	Equating unchanged ingestion with unchanged nutrition or reduced reproduction with reduced feeding.
Maintenance and reserves	Respiration, activity, ion balance, oxidative damage, ATP or metabolites, lipid quantity and reserve use.	Measure expenditure, reserves, feeding and production over matched intervals.	Inferring energetic benefit from biomarkers or lower respiration without functional endpoints.
Life history traits	Development, size, survival, egg number and quality, hatching, juvenile performance, sex ratio and population growth.	Cohort, reciprocal parent–offspring and multigenerational designs covering seasonal bottlenecks.	Inferring population resilience from one adult trait or generation.
Trophic transfer	Prey size spectrum, essential compounds per individual and volume, seasonal production, predator growth or survival, particle export.	Predator feeding trials or validated trophic models constrained by measured prey traits and fluxes.	Claiming effects on fish or food web production from zooplankton biomarkers alone.

These physiological changes must then be tracked across the life stages in which they affect population performance. Cohort studies should connect adult condition and reproductive allocation to egg quality, hatching success, juvenile survival, and the timing of recruitment. Exposure schedules should encompass seasonal transitions and periods of dependence on stored reserve. Reciprocal transfer experiments and common garden designs can help distinguish the effects of direct offspring exposure from those of parental exposure history. Genetic adaptation requires evidence linking selection or heritable genetic changes to improved performance, rather than relying on improved performance in later generations alone. Claims of epigenetic inheritance likewise require evidence that distinguishes inherited regulatory changes from the effects of continued exposure. Buffering in later generations should therefore be assessed alongside persistent fitness costs and performance under conditions different from those used during acclimation.

Field and recovery monitoring can help determine whether the mechanisms identified experimentally explain population persistence in natural systems. Measurements should characterize temporal variations in pH, food availability, temperature, and dissolved oxygen over biologically relevant periods, together with ionic conditions and ANC where they affect freshwater recovery. Consistent sampling habitats, depth, methods, and effort are needed to distinguish actual community changes from differences in sampling. Littoral

microcrustacean indicators can complement pelagic records in assessments of freshwater acidification [144]. Species recovery and functional richness should then be evaluated alongside grazing, production, and prey biochemical composition to determine how biological re-establishment relates to the restoration of nutritional function.

At the food web scale, studies should connect zooplankton production with the prey actually available to predators. Measurements should quantify prey size, essential compound content per individual, and seasonal prey availability during predator feeding periods. Predator feeding experiments that separate prey exposure from predator exposure can link prey characteristics to predator ingestion, assimilation, growth, and survival. Quantifying transfer efficiency requires clearly defined input and output fluxes measured over comparable time intervals. Nutrient recycling and particle export should be measured alongside trophic supply, as these processes may respond differently within the same community. The resource budget framework can inform the development of empirical models linking these processes, but reliable predictions require model parameterization and validation across different exposure regimes and food conditions.

9. Conclusions

Environmental acidification can alter zooplankton nutrition by changing both physiological demands and the food resources available to meet them. Because marine and freshwater acidification arise from different combinations of CO_2 enrichment, acid inputs and local biogeochemical processes, pH alone is insufficient to characterize comparable exposure conditions. Direct effects through physiological regulation, metabolic adjustment and cellular stress, and food-mediated effects through changes in prey availability, elemental balance and essential nutrients, can be linked within a resource budget framework of acquisition, assimilation, maintenance and allocation to growth and reproduction. Evidence from copepods and cladocerans shows that maintained adult traits can coexist with reduced resource conversion, reserve depletion, delayed development or impaired offspring performance. Adult tolerance therefore does not necessarily indicate maintained cohort-level production or population persistence. These responses vary with food supply, warming, contaminants and exposure history. Plasticity, epigenetic regulation and genetic adaptation provide only partial buffering across generations. As a result, the effects of acidification on prey quality, food web support and carbon cycling cannot be inferred from zooplankton abundance or biochemical composition alone, and the reviewed evidence includes neutral and positive as well as adverse responses. Because direct measurements of transfer from zooplankton to higher trophic levels remain scarce, the proposed trophic consequences should be regarded as testable pathways rather than established outcomes, and direct tests of transfer efficiency are a research priority. Quantifying resource conversion and stage-specific production through clearly defined trophic and ecosystem fluxes is needed to determine when compensation maintains ecological function and when nutritional costs persist despite apparently maintained performance.

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Abbreviations

The following abbreviations are used in this manuscript:

ANC	Acid-neutralizing capacity
CO_2	Carbon dioxide
DHA	Docosahexaenoic acid
DIC	Dissolved inorganic carbon
DOC	Dissolved organic carbon
FA	Fatty acid
PUFA	Polyunsaturated fatty acid
SSPs	Shared Socioeconomic Pathways
TA	Total alkalinity

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